PANCREATITIS—ITS TREATMENT, AS RELATED TO GALL-BLADDER INFECTION*

REPORT OF CASES

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SURGICAL diseases of the pancreas are not frequent. Unless a surgeon has seen several cases of acute pancreatitis, or unless he continually keeps this condition in his mind, he will fail to make a correct diagnosis before opening the abdomen. However, diseases of the pancreas associated with biliary tract disease are not so rare as formerly supposed.

INCIDENCE

In 1907 Egdahl reported a series of 105 cases of acute pancreatitis, forty-four of which were associated with gall stones. In a series of chronic pancreatitis cases reported by Deaver in 1921, 91 per cent showed evidence of biliary infection. In 1921 Judd found pancreatitis present in 26 per cent of 1290 cases in which operations were performed for biliary tract disease. In 1923 A. O. Whipple reported 230 cases of unselected biliary tract disease. Forty of these showed definite pancreatic changes. This emphasizes that in all operations in the upper abdomen, and especially on the biliary tract, the pancreas should be thoroughly examined.

The object of this paper is more definitely to crystallize our ideas about pancreatic lesions associated with biliary tract disease that our patients may be given full benefit from every surgical procedure. The surgical treatment must be based on the essential pathology.

ANATOMY AND PHYSIOLOGY

Let us consider the anatomy and physiology of the pancreas only as far as it has a bearing on the pathologic changes which take place.

The common bile duct is completely surrounded by the head of the pancreas in three out of four cases. In the fourth case the head of the pancreas is deeply grooved to receive the common duct. The pancreatic ducts usually empty with the common duct into the duodenum at the ampulla of Vater. They may, however, empty separately or may empty into the common bile duct one-third inch above the duodenum. As surgeons we are interested primarily in the external secretion of the pancreas.

The lymphatic supply, which is interstitial in distribution, drains toward the head of the pancreas and the common bile duct, and anastomoses with the lymphatics coming from the gall bladder. As shown by Graham and others, there is a close relationship between the lymphatics of the gall bladder, the liver, and the pancreas.

PATHOLOGY

Pancreatitis may be classified as acute or chronic with various in between stages.

Fitz, in his paper published in 1889, classified acute pancreatitis either as hemorrhagic, gangrenous, or suppurative. Acute pancreatitis is usually a necrotic process, often associated with hemorrhage when the necrotic process involves the blood vessels. The pancreas may be enlarged several times its normal size. It may be hard or edematous. It may be red or bluish black in spots or profuse, depending on the amount of hemorrhage present. The hemorrhagic type is often associated with fat necrosis and gall stones. The small yellowish white opaque areas of fat necrosis on the omentum and on the structures adjacent to the pancreas are quite unmistakable. In a large proportion of cases death occurs within the first four or five days. The gangrenous stage, characterized by discoloration and softening of necrotic tissue infiltrated with blood, represents a late stage of the same pathological process. Suppurative pancreatitis does not differ from similar lesions in other organs. Bacterial invasion may be caused by an extension from a suppurative inflammation of the bile ducts associated with gall stones, or it may follow a hemorrhagic necrosis of the pancreas. Fitz observed that fat necrosis is less frequently associated with suppurative inflammation than with hemorrhagic or gangrenous pancreatitis.

Chronic pancreatitis is chiefly characterized by an increase in the fibrous tissue of the pancreas. The surgeon is chiefly concerned with the interlobular type, which changes the character of the external secretion of the gland. At a late stage the contraction of the fibrous tissue causes the lobulations to become more distinct and to stand out as firm nodules. At a still later stage the entire gland feels like a hard nodular mass. This can be palpated at the time of operation. Not only may the fibrosis change the secretions of the pancreas, but due to the relation of the common bile duct, obstruction to the duct may result from the fibrosis and tumefaction of the head of the pancreas. Moynihan warns against making a diagnosis of chronic pancreatitis by palpating the gland alone, as variations in the normal are great. The possibility of the presence of carcinoma or syphilis must be excluded.

PATHOGENESIS

The cause of these pathologic changes in the pancreas is still a disputed question. The position of the gland, the rapid destructive changes, the masked symptoms, make accurate study most difficult.

Cases are on record where the infection has been traced to the appendix and to a duodenal ulcer. Evidence as to whether the infection or

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causal agent reaches the pancreas through the lymphatics or by way of bile retrojected into the ducts of the pancreas, is far from being conclusive.

Moynihan believes that the immediate cause of the necrosis in acute pancreatitis is the activation of the pancreatic secretion by some agent within the substance of the gland. In chronic pancreatitis he believes that infection by way of the lymphatics is the causative agent.

Archibald believes that altered bile retrojected into the ducts of the pancreas may activate the trypsinogen into active trypsin, thus leading to self-digestion of the pancreas. Such necrosis he believes may stop short at a lower level of destruction, resulting only in inflammatory reaction, the first stage of chronic fibrosis. He believes bacteria play only a secondary rôle and that gall stones and spasm of the sphincter of Oddi are mechanical factors.

The supporters of the lymphatic theory are equally emphatic in their beliefs. The frequent observation of a localized hepatitis about the gall-bladder bed, associated with cholecystitis and pancreatitis has given rise to the opinion that there is a close relationship between the gall bladder, liver, and pancreas. This view has been confirmed by the work of Graham. It is claimed that enlarged inflammatory lymph nodes about the base of the cystic duct and common duct may lead to a reversing of the lymphatic current and an infectious or noninfectious lymphangitis of the pancreas may result. Supporters of this theory believe that the chronic stage of pancreatitis is thus produced which may at any time activate the acute stage.

TREATMENT

Recovery from acute pancreatitis, except in the most severe types, may take place without operation. However, these cases are so rare that no case should be left unoperated.

Operative mortality for acute pancreatitis has decreased in direct ratio to the early diagnosis and prompt surgical treatment. The disease is most often mistaken for high intestinal obstruction or perforation of a peptic ulcer or of the gall bladder. Any one of these conditions requires prompt surgical intervention. Valuable time should not be wasted in making a differential diagnosis. The escape of pancreatic secretion and the extravasation of blood into the general peritoneal cavity is rapid. The abdomen is best opened through a paramedian incision above the umbilicus. When the abdomen is opened the escape of bloody fluid and the presence of areas of pancreatic fat necrosis make the diagnosis positive. However, hemorrhage may be extensive or very limited. It may be confined to the region of the pancreas or limited to its capsule. The pancreas may contain a small hemorrhagic area or the entire gland may be converted into a large red soft mass consisting of blood, fat necrosis, and parts of necrotic gland tissue. The bulging mass may crowd the stomach forward or appear through the gastrohepatic omentum, the gastrocolic omentum or the transverse mesocolon.

Upon opening the abdomen the bloody exudate should be aspirated as it is highly toxic. The general cavity should be well protected by gauze packs before proceeding to the exploration of the pancreas. The pancreas should be exposed by the most direct route, though the usual approach is through the gastrocolic omentum. The thin fibrous capsule of the pancreas may be slit with a curved forceps, care being taken not to plow into the gland, the purpose being to relieve the intraglandular tension. Vent must be given to any pent-up exudate, even if it is posterior to the pancreas. No effort should be made to loosen attached sloughs as hemorrhage may result. Abundant Penrose drains are then led down to contact with the pancreas. As infection of the biliary tract, with or without stones, so frequently coexists with acute pancreatitis, a careful examination of the gall bladder and ducts should precede the operation on the pancreas. If the gall bladder shows evidence of infection or stones, it should be drained whenever possible. If stones are in the common duct they should be removed and the duct drained. Drainage of the bile tract aids the escape of bile and prevents any increase of pressure within the ducts. Operative procedures undertaken in acute pancreatitis are emergency procedures and the least possible should be done. If the effusion into the general cavity is great, a suprapubic drain into the pelvis should be placed.

The postoperative treatment does not differ from that of any other acute abdominal condition. If the effusion has been extensive, the patient should be placed on a peritonitis routine with administration of 3000 to 4000 cubic centimeters of fluids a day by hypodermoclysis and proctolysis. The blood sugar should be watched. As in other conditions of lowered vitality the use of insulin and glucose may be indicated. The drain tubes should not be removed too early. Time must be given to have firm adhesions wall off the drainage tract and thus protect the general cavity against the digestive action of the secretions. The skin and abdominal wall may be protected by vaselined gauze, zinc oxid paste, or kaolin. The postoperative course is apt to be stormy.

Chronic pancreatitis is usually overlooked and is first recognized during the course of an operation on the biliary tract. In every case of chronic cholecystitis, chronic pancreatitis should be suspected. The first factor in treatment is removal of the cause of infection. This is necessary to prevent tissue changes from occurring in the pancreas which in time may cripple its function. Prompt and thorough removal of the primary focus of infection may arrest the disease before its more advanced form has developed. Drainage of the gall bladder, therefore, in chronic pancreatitis, is not a logical procedure. It does not do away with the infected gall bladder. Experience has shown that the patient is temporarily

benefited by gall bladder drainage, but recurrence of symptoms often occurs. Cholecystectomy, with drainage of the common duct, is recommended. In draining the common duct, Deaver and Judd use the T tube. The removing of the T tube may result in traumatism, which may lead to stricture of the common duct. Moynihan recommends the use of two catheters for drainage of the common duct. A large one, No. 10-14, is introduced into the hepatic duct, while a smaller one, No. 4-6, is inserted through the common duct into the duodenum. However, drainage of the gall bladder is justified in selected cases. In cases when the gall bladder does not seem diseased, and when there are no enlarged lymph nodes in the vicinity and the cause is elsewhere, as in a peptic ulcer, cholecystotomy has proved of great value. Doctor Lobingier has suggested leaving the stump of the gall bladder as a means of drainage. This removes the major part of the diseased gall bladder and avoids the hazard of any future stricture of the common duct. Drainage of the gall bladder or of the common duct, whichever is employed, must be continued over a long period of time.

A subacute stage of acute pancreatitis may result if the initial attack is less severe and if the resistance of the patient is sufficient to overcome the initial infection and shock. The whole attack is less severe. Where abscess and gangrene of the pancreas take place, early operation is indicated, as in the acute form. Usually, however, these are the cases seen late. The operative approach is the same as in acute pancreatitis. Drainage of the abscess must be made by the most direct route. Deaver has had occasion to drain these cases through the left lumbar route. When the lumbar drainage route is chosen, care must be used to avoid opening the peritoneum. It is best not to wipe out the abscess cavity with gauze as a severe hemorrhage may result. Abundant drainage is sufficient.

A pseudopancreatic cyst may be the result of acute hemorrhagic pancreatitis. It may be limited to the substance of the gland, or may be confined to the lesser peritoneal cavity. Prolonged drainage is indicated.

Following extensive destruction of the pancreas, diabetes mellitus may result. The only hope for such a patient is insulin. In diabetes, associated with gall-bladder disease, the source of infection should be removed. Marked improvement, and even cure, has been reported, especially in patients over forty years of age.

PANCREATITIS DEVELOPING AFTER OPERATIONS ON THE BILIARY TRACT

Some of the heretofore unexplained complications following operations on the biliary tract are now found due to acute pancreatitis. A so-called cardiac collapse occurring in the first twenty-four hours after operation may be due to pancreatitis. It may also occur later in the postoperative course. We should always bear this in mind in order to give our patients the benefit of proper drainage in such an event.

PANCREATIC ASTHENIA

Pancreatic asthenia is a symptom complex which may develop after any operation on the biliary tract, and especially when the pancreas and common duct are involved. A. O. Whipple, in 1923, was first to give an excellent description of this disease. The patient becomes listless, apathetic, develops nausea and vomiting, has a loathing for all food, complains of extreme exhaustion and weariness, and becomes highly apprehensive. There is a marked drop in blood pressure and a rapid loss in weight. These patients do not become comatose or delirious. All effort is avoided. All treatments are dreaded and exhausting. This group of symptoms usually comes on after the shock of operation has passed off and the patient is apparently doing well. It usually occurs between the second and ninth day postoperative, and may last two to thirty days if the patient survives.

Treatment should be directed chiefly to restore the body fluid loss. The continual vomiting and refusal to take fluids leads to low urinary output and nitrogen retention. Blood transfusions are indicated. Infusions of glucose, 5 to 10 per cent, with insulin have given excellent results. Digitalis has been given by rectum to improve the vasomotor and intestinal tone. Recovery, when such takes place, is said to be very rapid.

REPORT OF CASES

The following reports will illustrate the different types of pancreatitis I have encountered.

CASE 1.—Dr. C., age fifty-eight, was taken with severe pain in epigastric region about 3 a. m. The pain was most acute. The vomitus did not contain blood. The pulse was elevated to 112. He perspired freely, his respiration was 24. Later the pain was extended somewhat to right side of abdomen. The muscles of the abdomen were on guard, especially in the epigastric region, His past digestive history made us suspicious of gastric or duodenal ulcer. The history, the onset, and physical findings pointed to an acute condition within the abdomen, probably a perforated ulcer. Exploration was done within six hours from onset. This revealed the stomach and duodenum normal. There were adhesions about a distended gall bladder, but no perforation. The pancreas was enlarged; bloody fluid and flocculent lymph was in the lesser sac. There was no free fluid in general cavity and no fat necrosis. The fibrous capsule of pancreas was slit with a curved Kocher forceps. Several Penrose drains were led to the pancreas through an opening in the gastrocolic omentum. The gall bladder was drained and several stones removed. No stones were felt in common duct. The patient made an uneventful recovery though the convalescence was somewhat stormy. There is no doubt but that early operation stopped the acute pancreatitis in an early stage.

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CASE 2.—Mrs. W., age sixty-one, presented a cystic mass above umbilicus. Patient had had digestive disturbances, pain, nausea and vomiting for about four weeks. There was a history of attacks of gall-stone colic dating back three years. Exploration revealed the pancreas enlarged two to four times normal size. An abscess pointed between stomach, duodenum, and transverse colon. The lesser sac was obliterated by

adhesions. The gall bladder contained many stones. There were many adhesions about the above area. The abscess was drained. The stones were removed from the gall bladder and cholecystotomy done. The patient had a very stormy postoperative convalescence with severe abdominal pains and digestion of wound due to pancreatic secretions. Her ultimate recovery was satisfactory except for a postoperative ventral hernia.

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CASE 3.—Mrs. R., age fifty-three, a Mexican woman, was operated upon for acute cholecystitis and stone in common duct, with jaundice. Operation revealed a distended gall bladder, with thickened walls and filled with stones and pus. A marked perihepatitis about the gall bladder was present, with the liver edges rounded by swelling. The pancreas was enlarged, the lobules being prominent. The gall bladder was drained and the stones removed. The common-duct stone was removed and a No. 14 catheter anchored in the common duct, the tip placed in the direction of the hepatic duct. The postoperative course was satisfactory until the second week, when she developed the typical syndrome of pancreatic asthenia. She improved whenever the body fluids were restored, but would relapse as soon as they were stopped. This condition lasted for more than three weeks, when she died. No autopsy was permitted.

SUMMARY

In all operations in the upper abdomen the pancreas should be examined.

Operation for acute pancreatitis is done to help a patient over a serious emergency. No curative measures should be undertaken. The gall bladder should be drained. Not much time should be spent in removing stones.

In chronic pancreatitis cases the object should be to remove the focus of infection.

Postoperative complication of acute pancreatitis should be borne in mind in all operations on the biliary tract.

Pancreatic asthenia requires supportive treatment, restoration of body fluids, and the use of glucose and insulin.

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DISCUSSION

C. G. TOLAND, M. D. (1930 Wilshire Boulevard, Los Angeles).—Doctor Breyer has discussed the subject of acute pancreatitis from the standpoint of incidence, anatomy, physiology, pathogenesis, and treatment. I shall discuss the cause and the symptoms of acute pancreatitis.

The most serious of the acute abdominal diseases with which we, as physicians, have to deal is acute pancreatitis. The symptoms are acute, the diagnosis at times obscure, the treatment is radical, and the results are not entirely satisfactory. No one has been able definitely to prove the exact cause of acute pancreatitis and as a result there has appeared a large mass of experimental work and speculation on the subject. A number of men have produced acute hemorrhagic pancreatitis by injecting various substances, such as bile or duodenal contents into the pancreatic ducts, while others have worked out a lymphatic connection between the pancreas and appendix and gall bladder. They have shown that infection could extend from an acute process superimposed upon a chronic inflammation in either of these organs, through the retroperitoneal spaces to the pancreas.

Acute pancreatitis can be divided into three types: acute interstitial pancreatitis, acute suppurative pancreatitis or the pancreatic abscess, and the hemorrhagic type or pancreatic necrosis.

Acute pancreatic necrosis is generally considered to be a distinct disease and is rarely infectious in origin. The common cause is a retrojection of abnormal bile, that is, bile rich in salts, into the duct of Wirsung or to a retrojection of duodenal contents into the duct of Santorini. Either of these agents in the pancreatic ducts activates the proteolytic ferment, trypsin, resulting in digestion and necrosis of the parenchymatous cells, erosion of blood vessels, and hemorrhage.

Symptoms of Acute Pancreatitis.—Characterized by sudden severe pain in epigastrium radiating to the back, the left flank, to the lower chest, the left shoulder and over the entire abdomen, accompanied by abdominal distention, vomiting, and physical collapse. The extremities are blue, cold and clammy, and the patient is very restless.

Within twenty-four to forty-eight hours, if patient is alive, marked diarrhea begins. Tenderness is usually localized in the upper abdomen with marked resistance. At times a sausage-shaped mass may be felt in this same region. The temperature ranges from subnormal to 101, 102, or 103, and the white blood count is increased.

We must always keep in mind any previous history of gall stones, infected gall bladder, duodenal or gastric ulcer, appendicitis, or injury. The pain of an acute pancreatitis is not relieved by morphin so readily as in gall-stone colic. We must further differentiate incarcerated epigastric hernia; acute gastric dilatation; mineral poisoning; angina pectoris; spasm of the mesenteric arteries; mesenteric thrombosis; aneurysm of the abdominal aorta with slow leakage; intestinal obstruction; perirenal abscess of the left side; hematogenous infection of the left kidney; perforation of the undescended retrocolic appendix; ruptured ectopic pregnancy; and Pott's disease.

We will not discuss the matter of treatment except to emphasize Doctor Breyer's opinion that in acute pancreatitis immediate operation is necessary to save life.

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CLARENCE E. REES, M. D. (2001 Fourth Street, San Diego).—Acute pancreatitis when encountered by the surgeon usually presents the symptoms of a violent peritonitis. The patient is too ill to give a satisfactory history and the abdomen too rigid and tender for examination and localization of the primary focus. These patients are operated upon for acute surgical abdomen with a usual diagnosis of ruptured viscus; the pancreatic origin usually constitutes a surgical surprise. The hemorrhagic peritonitis, with the milk-white areas of fatty necrosis, should lead one directly to the diseased pancreas the treatment of which has been well outlined. Any prolonged procedure on bile tracts other than cholecystostomy should be avoided as these patients are poor risks. Sufficient drainage of the abdomen and postoperative support have been stressed.

In other abdominal operations the pancreas should be handled with the utmost care, as a postoperative acute pancreatitis is a fatal complication. Such a complication is particularly likely to occur in operations on the common bile duct, where a stone is encysted at the ampulla of Vater. Attempts to elevate the stone from the ampulla by stripping and manipulating the duct in the region of the pancreas, if made at all, should be made with the utmost care as this is probably the most frequent cause of surgical pancreatitis. Also in transduodenal removal of calculi from the common duct the ampulla should be definitely isolated and enlarged only enough to permit removal of the stone. Incision of the common duct through the duodenum in the region of the stone rather than through the ampulla is a much easier procedure but is attended by a much higher mortality because it permits a retroperitoneal leak of pancreatic secretion.

When the abdominal drainage is irritating and contains pancreatic ferments the skin in the region of

the wound can be well protected with a dried milk dressing.* Dried milk is sprinkled thickly on the skin around the drainage tubes, a layer of gauze placed over this and milk again sprinkled over the gauze; the usual dressings are then applied. Dried milk, with its high concentration of fat, protein, and carbohydrate, has the advantage of neutralizing all of the ferments of the pancreas and thus aids in protecting the skin.

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CHARLES A. DUKES, M. D. (426 Seventeenth Street, Oakland).—Pancreatitis is a disease which has been frequently overlooked not only by the surgeon but by the diagnostician, especially the type of cases which have been reviewed under the head of subacute pancreatitis and those which follow operation on the biliary tract.

In the treatment of an acute condition of the abdomen where the diagnosis or operation seems somewhat hazy, where the appendix is not sufficient to account for the acute condition and the gall bladder is not sufficiently involved to lead one to suspect this as a cause of the condition, the surgeon certainly is negligent who does not expose the pancreas and definitely determine its condition.

It has seemed to me at times that, in the severe conditions which accompany acute pancreatitis, we are prone to use a type of anesthetic that does not give complete relaxation and the best opportunity for exploration of the abdomen. In these acute conditions there is a natural tendency to hurry the investigation.

I am very much impressed with the necessity for thorough drainage in these cases. I think this has been excellently shown in a paper by Olds, read before this section last year. There is no doubt in my mind that proper drainage of these cases will further reduce the mortality rate.

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DOCTOR BREYER (Closing).—Because of lack of time it was not possible for me to take up the symptomatology of pancreatitis. I am very glad that the discussers have emphasized the difficulty of making an accurate diagnosis. We shall be glad to try on our next case the dried milk as a dressing for intestinal fistula suggested by Dr. Clarence E. Rees.

POSTOPERATIVE MASSIVE ATELECTASIS†

A DISCUSSION OF ITS ETIOLOGY, PREVENTION AND TREATMENT, WITH REPORT OF CASES

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DISCUSSION *by Frederick Leet Reichert, M. D., San Francisco.*

ALTHOUGH the clinical aspects of postoperative massive collapse have become thoroughly familiar to surgeons through the recent reports of Scrimger,[1] Scott,[2 3 4] Churchill,[5] Jackson,[6] Lee,[7] Sante,[8] et al., the etiology of this most interesting phenomenon remains sufficiently obscure to warrant the detailed presentation of individual experiences which provide important and possibly illuminating evidence as to the mechanism of its production.

PATHOLOGY

In his original definition of postoperative atelectasis William Pasteur emphasized "the failure of inspiratory power" as being responsible for the sudden deflation of large areas of lung tissue, a deflation which he thought occurred in the absence of any signs of obstruction of the airways. The failure in inspiratory power he attributed to the loss of the mobility of the diaphragm, either by direct paralysis or by reflex inhibition from acute inflammation. Briscoe attributes the deflation of the lung partly to the normal consequence of diminished breathing incident to a prolonged supine posture, incident to a severe illness, and partly to the alterations in the action of the diaphragm and of the muscles accessory to the diaphragm produced by inflammation of the muscles or of the pleural membrane covering them.

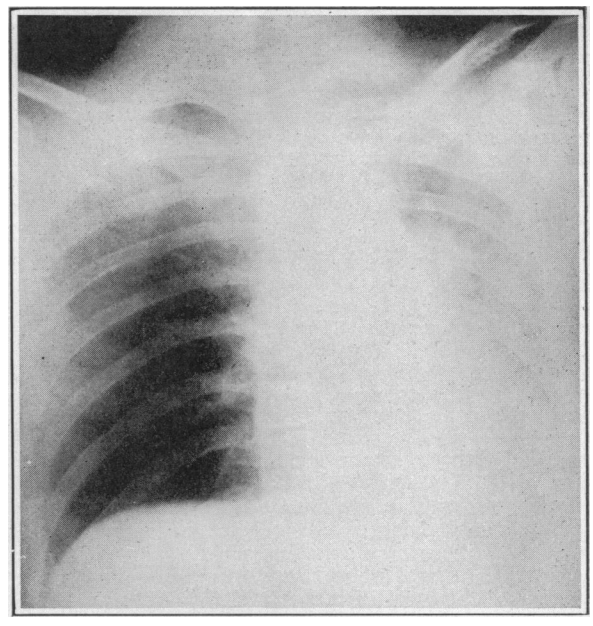

Fig. 1.—Massive atelectasis of the left lung with displacement of the mediastinum and heart to the left fourteen hours after appendectomy. Case 2.

Most modern authors consider that bronchial obstruction associated with a weakened or diminished respiratory force plays the important part in the development of atelectasis. The site of this bronchial obstruction was thought by Elliott and Dingley[9] and later by Scott,[2] to be located in the bronchioles and peripheral respiratory passages, whereas Lee and Jackson[6] consider plugs of thick, tenacious mucus in the larger bronchi, observable and removable by bronchoscope, to be responsible for the obstruction.

To explain the hypothetical obstruction in the bronchioles, Scott suggests that the fundamental condition which initiates massive atelectasis is a nervous reflex, probably vasomotor, which causes a bilateral, partial obstruction in the peripheral respiratory passages, and that posture and tenacious sputum are secondary factors which make this obstruction complete on one side in advance of the other with a subsequent unilateral absorption of air to complete the picture of

* Rees, Clarence E.: Dried Milk as a Dressing for Intestinal Fistula, California and West. Medicine, 30:419, 1929.

† Read before the Anesthesiology Section of the California Medical Association at the Fifty-Eighth Annual Session, May 6-9, 1929.